Supplementary Materials

Selection of Studies

The BrainMap database was employed for the retrieval of relevant neuroimaging experiments. As reported in the User Manual, BrainMap uses a structured standardized coding scheme which describes published human neuroimaging experimental results. This taxonomy has been used to describe over 3600 publications and 15000 experiments, drawing upon over 110000 subjects and reporting over 120000 coordinates. The main division of the coding scheme is between *structural* (VBM) and *functional* data. For this meta-analysis only papers labeled as "Structural" have been used. Considering the studies in this category, the database consists of 980 papers, 3093 experiments, 73938 subjects and 21481 locations.

The software application "Sleuth" has been used to search the database for experiments of interest and view the relevant search results in a standard brain space. This procedure allowed us to identify 242 studies involving SCZD, ASD, and OCSD (126, 96 and 20, respectively).

In a second step, a systematic search strategy was used to identify relevant studies whose temporal boundaries are not included in the BrainMap database, published until 15 July 2016, across the online database most frequently used in the international literature (Medline database with PubMed literature search: http://www.ncbi.nlm.nih.gov/pubmed) involving SCZD, ASD, and OCSD as they are defined by DSM-5 (American Psychiatric Association, 2013).

First, association measures have been analyzed to get a perspective on the biomedical research literature: co-occurrences of all the terms concerning psychiatric spectra and neuroimaging acquisition technique have been analyzed by measuring the degree to which two queries coincide among all publications. The corresponding co-occurrences of publications have been returned in the form of natural logarithm of the Jaccard index (ln J) (Jaccard, 1901). Secondly, we have adopted the MeSH hierarchy for Mental Disorders in PubMed automatic routines to find relevant published article concerning ASD, SCZD and OCSD. In particular, we provided a *set* of PubMed queries as input, adopting Boolean operators and PubMed search field tags. Indeed, to identify the greatest number of items we used the function [ALL] concerning the possibility to find MeSH terms in "all field" of an article. For each spectrum, the search algorithm has been constructed as follows:

- FIRST SET
    - (#spectrum 1: "autism spectrum disorder" OR [ALL] "ASD" [ALL]) AND (# acquisition technique: "voxel-based morphometry" [ALL] OR "VBM" [ALL]; "diffusion tensor imaging" OR "DTI" [ALL]).
- SECOND SET
    - (#spectrum 2: "obsessive-compulsive disorder" OR [ALL] "OCD" [ALL]) AND (# acquisition technique: "voxel-based morphometry" [ALL] OR "VBM" [ALL]; "diffusion tensor imaging" OR "DTI" [ALL]).
- THIRD SET

- (#spectrum 3: “schizophrenia” OR [ALL] “schizoaffective disorder” [ALL]) AND (# acquisition technique: “voxel-based morphometry” [ALL] OR “VBM” [ALL]; “diffusion tensor imaging” OR “DTI” [ALL]).

As regards the search and selection process, additional considerations are necessary: (1) concerning the OCSD, PubMed has found under the heading of the spectrum also articles about Tic disorder that, after a manual verification, were found to contain the correct MeSH term of classification [i.e., Study ID: 12,13,31,50,51,71]; (2) at the beginning of the data collection in 2012, PubMed did not contain a MeSH term for SCZD: so it has been necessary to resort to the terms “schizophrenia” and “schizoaffective disorder”.

Up until 15 July 2016, 1419 papers involving SCZD, ASD, and OCSD had been indexed on BrainMap database and PubMed. All the selected articles that did not meet the inclusion criteria were excluded. In particular, two experienced researchers have reviewed all the articles using a double-blind procedure to ensure: (1) both the presence of the healthy control group and the pathological sample; (2) that the results were reported by using the Talairach/Tournoux or Montreal Neurological Institute (MNI) coordinates; (3) that the foci of interest had a significance of at least <0.05; (4) that the studies described cerebral structural changes visible with VBM or DTI (only FA technique); (5) that the studies were original works; (6) that original diagnosis was made on the basis of DSM criteria and clinical test batteries. Furthermore, instances of multiple references to the same datasets across articles were identified so as to make sure that only one reference to the same data contributed to the coordinates for the present meta-analysis (See Graph S1 [PRISMA flow chart] and Table S1).

All the studies were examined independently to detect dissimilarities or discrepancies, which were afterward collectively discussed and resolved. The researchers who carried out this research stage have reached substantial agreement (% of agreement = 94.3096; Cohen’s K= 0.7409).

Relevant descriptive information was extracted from each article. On the grounds of the aforementioned criteria, 203 papers were included in the meta-analysis, with 218 identified experimental samples and a total of 8693 subjects: 1719 individuals of the ASD group, 1738 of the OCSD group, and 5236 of the SCZD group. Tables S1-S2 provide a detailed description of methods and the sample of the selected studies (see also Figure S2). The number of WM and GM changes foci were established for each study. In order to facilitate analysis, coordinates from MNI space were converted into Talairach coordinates by using Lancaster transformation (Lancaster et al., 2007).

Supplementary Tables and Figures

Figure S1. Selection strategy.

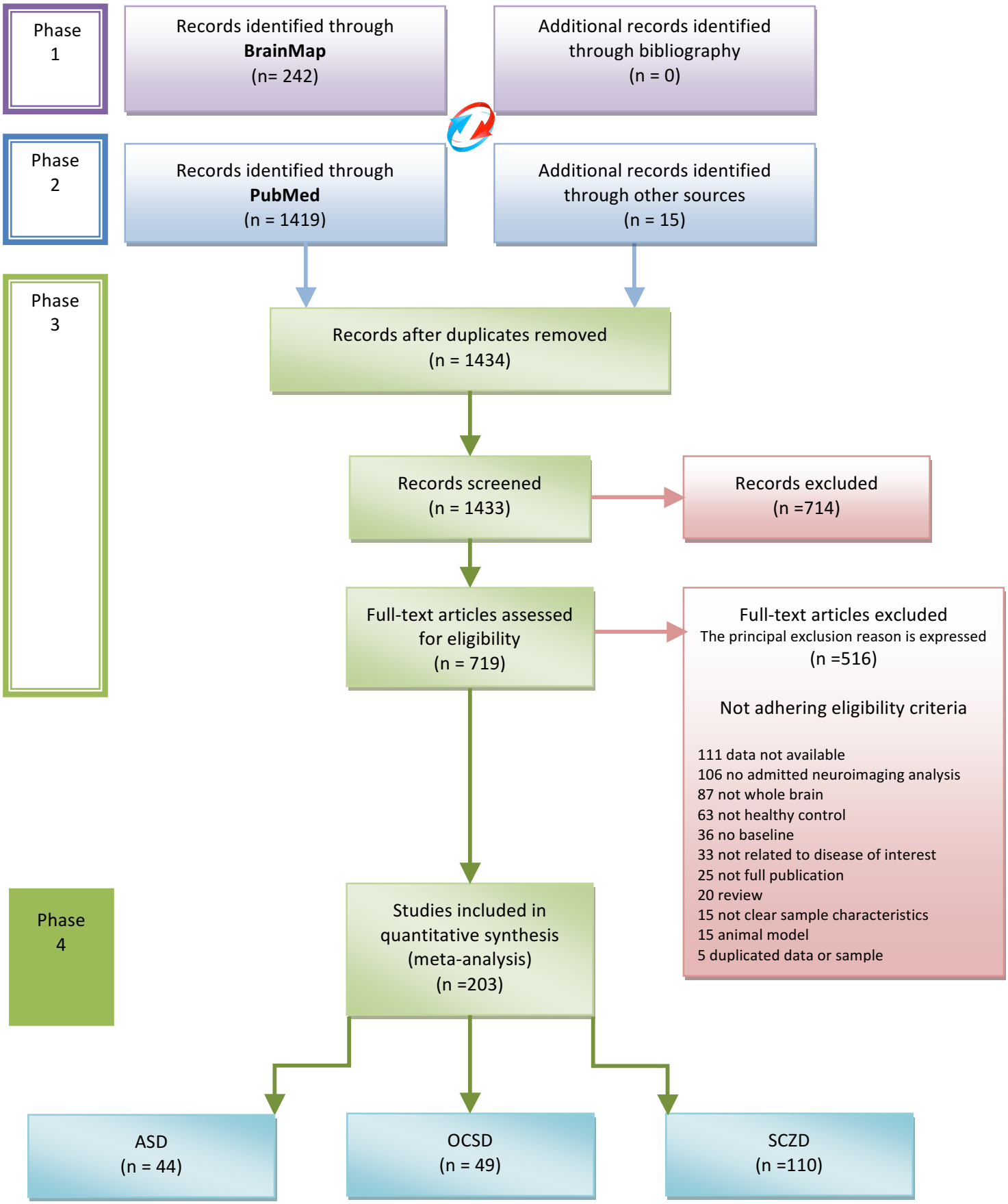


Table S1. Selected studies for the meta-analysis[1].

ID		First Author	Year	Reference	N	M/F	age (year)*	Scanner (Tesla)	slice thick (mm)**	Smoothing (mm)	Method
1	ASD	Cheung C	2009	Jour Child Psychology Psychiatry 50(9): 1102-12	13	12/1	9.3±2.6	1.5	5	6	SPM2
2	ASD	Cheng Y	2010	Neuroimage 50(3): 873-82	25	25/0	13.71±2.54	1.5	2.2	-	FSL4.1
3	ASD	Noriuchi M	2010	Brain Research 1362: 141-9	7	6/1	13.96±2.68	3	2	6	IDL/ SPM2
4	ASD	McAlonan GM	2002	Brain 125: 1594-1606	21	19/2	32±10	1.5	1.5	-	SMaRT on SPARC
5	ASD	Craig MC	2007	Brit Jour Psychiatry 191: 224-8	14	0/14	37.9±11.4	1.5	1.5	5	SPM2
6	ASD	McAlonan GM	2009	Psychol Med 39(11): 1885-93	18 18	15/3 15/3	11.2±2.5 11.6±3	1.5	3	4.4	BAMM on SPARC workstation
7	ASD	McAlonan GM	2005	Brain 128(Pt 2): 268-76	17	16/1	12±1,8	1.5	3	4.4	BAMM on SPARC workstation
8	ASD	Mengotti P	2011	Brain Res. Bull. 84(2): 189-95	20	18/2	7±2.25	1.5	5	8	SPM5
9	ASD	Spencer MD	2006	Neuroimage 33(4): 1136-44	63	34/29	16.0±1.8	1.5	-	12	SPM99
10	ASD	Ecker C	2010	Neuroimage 49(1): 44-56	22	22/0	27±7	3	1.1	8	SPM5
11	ASD	Ke X	2009	Brain Research 1265: 171-7	12	12/0	11.2±2.5	1.5	2	8	SPM5
12	OCSD	Mueller-Vahl KR	2009	BMC Neuroscience 10: 47	19	19/0	18-60	1.5	3	8	SPM2
13	OCSD	Thomalla G	2009	Brain 132: 765–777	15	13/2	34.5±8.9	3	1	8	SPM2
14	OCSD	Lochner C	2012	Jour Psychiatry Neurosci 37(3): 193-9	15	10/5	11.2±4.9	3	2.2	-	FSL-TBSS
15	OCSD	Menzies L	2008	Am J Psychiatry 165: 1308–1315	30	9/21	32.2±11.1	1.5	4	8	SPM5
16	OCSD	Yoo SY	2007	Acta Psychiatr Scand 116: 211–219	13	8/5	27.8±7.3	1.5	-	10	SPM2
17	OCSD	Togao O	2010	Psychiatry Research: Neuroimaging 184: 29–37	23	9/14	32.6±9.7	1.5	-	12	SPM2
18	SCZD	Whitford TJ	2007	Am Journ Psychiatry 164: 1082–1089	41	-	19.8±3.3	1.5	-	12	SPM2
19	ASD	Barnea-Goraly N	2004	Biol Psychiatry 2004 Feb 1;55(3):323-6	7	7/0	14.6±3.4	1.5	5	4	SPM99
20	ASD	Keller TA	2007	Neuro Report 18(1): 23-7	34	34/0	18.9±7.3	3	3	8	SPM2
21	ASD	Cheon KA	2011	Brain Research 1417: 77-86	17	17/0	11.0±2.1	1.5	3	_	DTIfit/FSL TDT toolbox
22	ASD	Jou RJ	2011a	Austr New Ze Jou Psychiatry 45(2): 153-62	10	10/0	13.5±4	1.5	4	_	BioImage Suite

[1] The items shown in the table are the result of the entire selection process as shown in PRISMA (2009) flow chart and table S2. The starting point for the selection can be traced in the algorithms and in the additional considerations previously proposed.

23	ASD	Jou RJ	2011b	AJNR Am Jour NeuroRadiology 32(9): 1607-13	15	-	10.9±3.7	3	2.5	-	FSL/TBSS
24	ASD	Pardini M	2009	Eur Journ Neurol 16(11): 1185-90	10	10/0	19.7±2.83	3	2	4	FDT
25	ASD	Toal F	2010	Psychol Med 40(7): 1171-81	65 39 26	56/9 35/4 21/5	31±10 32±12 30±8	1.5	-	8	SPM2
26	ASD	Boddaert N	2004	Neuroimage 23(1): 364-9	21	16/5	9.3±2.2	1.5	1.2	12	SPM99
27	ASD	Bonilha L	2008	Brain Dev 30(6): 396-401	12	12/0	12.4±4	2	-	10	SPM5
28	ASD	Hyde KL	2010	Human Brain Map 31(4): 556-66	15	15/0	22.7±6.4	3	-	12	CIVET
29	ASD	Waiter GD	2005	Neuroimage 24(2): 455-61	15	15/0	15.2±2.2	1.5	-	8	SPM2
30	ASD	Ke X	2008	Neuroreport 19(9): 921-5	17	14/3	8.88±1.96	1.5	2	8	SPM5
31	OCSD	Neuner I	2010	NeuroImage 51(3): 1184-93	28	20/8	30.05±10.78	1.5	2	-	FMRIB
32	OCSD	Chamberlain SR	2010	Arch Gen Psychiatry 67(9):965-71	18	1/17	37.39±11.65	1.5	4	-	FMRIB
33	OCSD	Qing Fan	2012	PLoS ONE 7(4): e35889	27	17/10	25.5±7	1.5	5	6	SPM8
34	OCSD	Nakamae T	2008	Progress in Neuro-Psychopharmacology & Biological Psychiatry 32: 1221-226	15	9/6	29.7±6.9	1.5	-	-	SPM2
35	OCSD	Szeszko PR	2005	Arch Gen Psychiatry 62: 782-790	15	10/5	38.5±10.9	1.5	5	-	FSL
36	OCSD	Lazaro L	2011	Progress in Neuro-Psychopharmacology & Biological Psychiatry 35: 1863–1869	27	15/12	15.6±1.5	1.5	1.5	12	SPM5
37	OCSD	Lazaro L	2009	Psychiatry Research: Neuroimaging 172: 140–146	15	8/7	13.7±2.5	1.5	1.5	12	SPM5
38	OCSD	Duran FL	2009	Neuroscience Letters 452: 68–71	19	10/9	32.7±8.8	1.5	1.2	12	SPM
39	OCSD	Van Den Heuvel OA	2009	Brain: a journal of neurology 132: 853-868	55	16/39	33.7±9.19	1.5	1.5	12	SPM5
40	OCSD	Carmona S	2007	Neuroscience Letters 421: 218–223	18	13/5	12.86±2.76	1.5	2	8	SPM2
41	SCZD	Oertel-Knöchel V	2012	Schizophrenia Research 138: 120-127	31	16/15	38 ±11.24	3	1	8	SPM8
42	SCZD	Chua SE	2007	Schizophrenia Research 89: 12–21	29	12/17	32±10	1.5	3	4.4	BAMM
43	SCZD	Ananth H	2002	Am Journ Psychiatry 159(9): 1497-505	20	10/10	37.8±9.5	2	1.5	8	SPM99
44	ASD	Brieber S	2007	Jou Child Psychol Psychiatry 48(12):1251-8	15	-	14.2±1.9	1.5	-	12	SPM2
45	ASD	Rojas DC	2006	BMC Psychiatry 6: 56	24	24/0	22.6±11.61	1.5	-	8	SPM2
46	ASD	Waiter GD	2004	Neuroimage 22(2): 619-25	16	16/0	15.4±2.24	1.5	-	8	SPM2
47	ASD	Salmond CH	2005	Eur Jour Neurosci 22(3): 764-72	14	13/1	12.9±0.7	1.5	-	12	SPM99
48	ASD	Calderoni S	2011	Neuroimage 59(2): 1013-22	38	0/38	4.4±1.5	1.5	1	8	SPM8
49	ASD	Schmitz N	2006	Biol Psychiatry 59(1): 7-16	8	8/0	38±9	1.5	-	10	SPM99
50	OCSD	Ludolph AG	2006	British Journal of Psychiatry 188: 484-485	14	14/0	12.5	1.5	-	-	SPM2

51	OCSD	Garraux G	2006	Ann Neurol 59: 381–385	31	25/6	32±10.5	3	1.3	8	SPM2
52	OCSD	Zarei M	2011	BIOL PSYCHIATRY 70: 1083–1090	26	14/12	16.6±1.5	1.5	1	3	FMRIB
53	OCSD	Christian JC	2008	Psychiatry Research: Neuroimaging 164: 123–131	21	15/6	38±9.6	1.5	1.5	8	SPM2
54	OCSD	Szeszko PR	2008	Am Jour Psychiatry 165: 1299–1307	37	14/23	13±2.7	1.5	1.5	8	SPM2
55	OCSD	Gilbert AR	2008a	Journal of Affective Disorders 109: 117–126	25	13/12	37.5±10.7	1.5	1.5	12	SPM5
56	OCSD	Gilbert AR	2008b	Neuroscience Letters 435: 45–50	10	6/4	12.9±2.7	1.5	1.5	8	SPM2
57	OCSD	Yoo SY	2008	Journ Korean Med Sci 23: 24-30	71	47/24	26.61±7.5	1.5	-	12	SPM2
58	OCSD	Valente AA	2005	BIOL PSYCHIATRY 58: 479–487	19	10/9	32.7±8.8	1.5	1.2	8	SPM2
59	SCZD	Yüksel C	2012	Schizophrenia Research 138: 177-182	43	28/15	38.7±10.6	3	1.33	12	FSL-VBM
60	SCZD	Horacek J	2011	The World Journal of Biological Psychiatry 13(7): 501-9	44	22/22	30.82±9.76	1.5		8	SPM5
61	SCZD	Aleksic B	2012	Schizophrenia Bulletin 39(3):720-8	100	53/47	38.3±13	1.5	1.4	12	SPM5
62	ASD	Kwon H	2004	Dev Med Child Neurol 46(11): 760-4	11	11/0	13.6±2.4	3	-	-	SPM99
63	ASD	McAlonan GM	2008	Jou Child Psychol Psychiatry 49(12): 1287-95	17 16	14/3 14/2	7-16	1.5	3	4.4	BAMM on SPARC workstation
64	ASD	Kurth F	2011	Biol Psychiatry 70(3): 278-82	52	38/14	11.2±3.95	1.5	1.2	8	SPM8
65	SCZD	Federspiel A	2006	Neurobiol Dis 22: 702-709	12	8/4	23.4±3	1.5	-	7.5	-
66	ASD	Salmond CH	2007	Cortex 43(6): 686-99	26	-	8-18	1.5	-	12	SPM99
67	ASD	Wilson LB	2009	Psychiatry Research 174(2): 138-145	10	8/2	30.1±9.18	1.5	1.7	-	SPM2
68	SCZD	Shin YW	2006	Neuroimage 30(4): 1285-91	19	11/8	27.84±4.78	1.5	-	-	SPM2
69	ASD	Kosaka H	2010	Neuroimage 50(4): 1357-63	32	30/2	23.8±4.2	3	-	-	SPM5
70	ASD	Riva D	2011	AJNR Am Jou Neuroradio 32(8): 1430-5	21	13/8	6.6±2.5	1.5	-	8	SPM5
71	OCSD	Wittfoth M	2012	BMC Neuroscience 13: 17	29	29/0	30.7±9	1.5	-	8	SPM8
72	OCSD	Matsumoto R	2010	Psychiatry and Clinical Neurosciences 64: 541–547	16	7/9	32.8±7.5	1.5	1	12	SPM5
73	OCSD	Koprivovà J	2009	Neuroscience Letters 464: 62–66	14	5/9	28.6±6.1	3	-	10	SPM5
74	OCSD	Cecconi JP	2008	Neuroscience Letters 447: 138–142	5	2/3	35±11.07	1.5	1.56	8	SPM2
75	SCZD	Ferri F	2012	Neuropsychologia 50: 988-996	19	14/5	27.2±5.4	1.5	-	8	SPM8
76	SCZD	Chow EWC	2011	Am J Psychiatry 168(5): 522-529	29	11/18	30.7±8.5	1.5	1.5	12	SPM5
77	SCZD	Donohoe G	2010	NeuroImage 54(3):2132-7	70	46/24	40.44±11.7	1.5	1	8	SPM5
78	SCZD	Venkatasubramanian G	2010	Indian J Psychiatry 52: 28-36	30	21/9	30.1±8.3	1.5	1	8	SPM2
79	SCZD	Pomarol-Clotet E	2010	Molecular Psychiatry 15: 823-830	32	21/11	41.56±8.79	1.5	-	4	FSL-VBM

80	SCZD	Euler M	2009	Schizophr Res 115(1) :1-7	20	18/2	43.26±10.5	1.5	1.5	12	SPM5
81	SCZD	Voormolen EH	2010	Neuroimage 49(1):587-96	14	7/7	28±6.4	1.5	-	12	SPM5
82	SCZD	Horn H	2010	Psychiatric Research: Neuroimaging 182: 183-186	20	13/7	30.1±10	1.5	1	10	SPM5
83	SCZD	Lui S	2009	American Jour Psychiatry 166 (2): 196-205	68	30/38	24.2±8.6	3	1	8	SPM2
84	SCZD	Voets NL	2008	NeuroImage 43(4): 665-75	25	18/7	16±1.4	1.5	-	8	FSL
85	SCZD	Meda SA	2008	Schizophr Res 101(1-3): 95–105	200	112/88	39.69±12.06	1.5	1.5	8	SPM2
86	SCZD	Xu L	2009	Hum Brain Mapp. 30(3): 711–724	120	69/51	42.1±12.9	1.5	1.5	12	SPM5
87	SCZD	Shaufelberger MS	2007	British Journal of Psychiatry 191: s117-s122	62	44/18	27.6±8	1.5	-	8	SPM2
88	SCZD	Garcìa-Martì G	2008	Progress in Neuro-Psychopharmacology & Biological Psychiatry 32: 72–80	17	17/0	35.71±6.11	1.5	1.25	12	SPM2
89	SCZD	Douaud G	2007	Brain; 2007 Sep; 130(Pt 9):2375-86	25	18/7	13-18	1.5	1	8	FSL FMRIB
90	SCZD	Martì-Bonmatì L	2007	Radiology: Volume 244: Number 2 August 2007	21	-	39±10	1.5	1.25	8	SPM2
91	SCZD	Kubicki M	2002	Neuroimage. 2002 December; 17(4): 1711–1719.	16	14/2	26±7.5	1.5	-	12	SPM99
92	SCZD	Janssen J	2008	J. Am. Acad. Chil Adolesc. Psychiatry 47(11):1311-20	25	19/6	15.4±1.8	1.5	1.5	12	SPM2
93	SCZD	Tang J	2012	PlosOne 7: e40247	29	16/13	16.5±0.9	1.5	1.8	8	SPM5
94	SCZD	Benoit A	2012	Front Psychiatry 3: 42	16	13/3	24.2±4,3	1.5	1	10	VBM8
95	SCZD	Shergill SS	2007	American Journ of Psychiatry 164(3): 467-73	33	30/3	32±10	1.5	-	4	FMRIB
96	SCZD	Rametti G	2010	Schizophrenia Research 121: 66-74	23	11/12	32.1±7.1	1.5	1.5	8	SPM5
97	SCZD	Herbsman T	2010	Schizophr Research 116(1): 99-100	13	-	37.4	3	3	-	FSL
98	SCZD	Knöchel C	2011	Neuroimage 59(2): 926-34	16	9/7	37.57±7.84	3	1	-	FSL
99	SCZD	Pagsberg AK	2007	Journal of Neural Transmission 114: 489-498	29	-	15.7±1.8	1.5	-	12	SPM99
100	SCZD	Hubl D	2004	Arch Gen Psychiatry 61(7): 658-68	13 13	8/5 8/5	33.8±1.5 31±9.3	1.5 1.5	1.2 1.2	- -	- -
101	SCZD	Szeszko PR	2005	American Journ of Psychiatry 162(3): 602-5	10	6/4	26.9±4.6	1.5	1.5	3	FSL
102	SCZD	Buchsbaum MS	2006	Biol Psychiatry 60(11): 1181-7	63	44/19	41.7±12.5	3	3	-	AIR5.1
103	SCZD	Hao Y	2006	NeuroReport 17(1): 23-26	21	12/9	23.71±5.47	1.5	4	8	SPM2
104	SCZD	Ashtari M	2007	Arch Gen Psychiatry 64(11): 1270-80	23	13/10	15.8±1.9	1.5	2.5	-	DTIStudio
105	SCZD	Manoach DS	2007	Neuroimage 37(2): 599-610	17	13/4	41±12	3	2	-	-
106	SCZD	Mori T	2007	Psychiatry Research 154(2): 133-45	42	26/16	40±9.3	1.5	1.23	-	SPM2
107	SCZD	Schlösser RG	2007	Schizophrenia Research 89(1-3): 1-11	18	14/4	29.6±7	1.5	3	8	SPM2
108	SCZD	Cheung V	2008	Psychological Medicine 38(6): 877-85	25	11/14	18-45	1.5	3	6	SPM2

109	SCZD	Kyriakopoulos M	2008	Biol Psychiatry 63(5): 519-23	19	12/7	17.09±1.69	1.5	2.5	4	SPM2
110	SCZD	Bai YM	2009	Schizophrenia Research 109 (2009): 167-181	20	5/15	40.5±9.3	1.5	2.2	8	SPM2
111	SCZD	Hao Y	2009	Schizophrenia Research 114(1-3): 128-35	34	20/14	25.44±5.90	1.5	4	8	SPM2
112	SCZD	Hashimoto R	2009	World Journ Biolog Psychiatry 10(1): 65-9	42	26/16	40±9.3	1.5	1.23	6	MATHLAB 6.5
113	SCZD	Kanaan R	2009	British Journal Psychiatry 194(3): 236-242	76	66/10	30.9±10.2	1.5	2.5	8	SPM2
114	SCZD	Rotarska-Jagiela A	2008	NeuroImage 9(4): 1522–1532	24	12/12	39±9.35	3	-	-	-
115	SCZD	Sussmann JE	2009	Bipolar Disorders 11(1): 11-18	28	15/13	38±9.9	1.5	2.8	8	SPM2
116	SCZD	Chan WY	2010	Schizophrenia Research 119 (1-3): 52–60	39	30/9	28.8±6.8	3	0.9	-	SPM5
117	SCZD	Perez-Iglesias R	2010	Am J Psychiatry 167(4): 451-8	49	-	-	1.5	1.5	4	SPM2
118	SCZD	Tang J	2010	Brain Research 9 July 2010, Pages 199–205	38	20/18	16.3±1	1.5	4	8	SPM5
119	SCZD	Nakamura K	2012	Psychiatry Research: Neuroimaging 202(3): 233–238	58	38/20	27,6±6.9	1.5	5	-	SPM2
120	SCZD	Sugranyes G	2012	Schizophrenia Research 138(2-3): 136–142	22	14/8	17.1±1.5	1.5	-	-	SPM5
121	SCZD	Cui L	2011	Psychiatry Research: Neuroimaging 194: 347-353	25	16/9	25.8±6	3	-	-	SPM2
122	SCZD	Colombo RR	2012	Psychiatry Res 2012 202(3): 198-205	62	45/17	27.74±8	1.5	-	12	SPM2
123	SCZD	Hulshoff Pol HE	2004	NeuroImage 21: 27-35	159	112/47	35.6±12.4	1.5	1.2	8	-
124	SCZD	Honea RA	2008	Biological Psychiatry (2008) 63 465-474	169	132/37	36.39±9.46	1.5	1.5	8	SPM2
125	SCZD	Kawasaki Y	2004	Eur Arch Psychiatry Clin Neurosci 254: 406-414	25	14/11	25.8±4.5	1.5	1	12	SPM99
126	SCZD	Manè A	2009	Schizophrenia Research 114: 136-143	15	12/3	25.56±5.77	1.5	-	-	SPM5
127	SCZD	Hulshoff Pol HE	2001	Arch Gen Psychiatry 58: 1118-1125	159	112/47	35.6±12.4	1.5	1.2	8	-
128	SCZD	Van Haren NEM	2007	Neuropsychopharmacology 32: 2057–2066	96	70/26	32.22±11.10	1.5	-	8	-
129	SCZD	Shiffer B	2010	Brain: A Journal of Neurology 133: 3093-3103	24	24/0	37.55±8.45	1.5	1	14	SPM5
130	SCZD	Segall JM	2009	Schizophrenia Bulletin 35(1): 82-95	266	167/99	33.7±11.7	MULTICENTRE			
131	SCZD	Cooke MA	2008	Schizophrenia Research 103: 40-51	52	40/12	38.35±9.89	1.5	5	8	SPM2
132	SCZD	Prasad KMR	2007	Molecular Psychiatry 12: 105-113	30	23/7	24.66±7.71	1.5	1.5	12	SPM2
133	SCZD	Koo MS	2006	Arch Gen Psychiatry 63: 1090-1100	30	0/30	29.8±9.4	1.5	1.5	8	SPM2
134	SCZD	Nenadic I	2011	Schizophr Bull 38(4): 838-44	99	57/42	36.2±11.2	1.5	1x1x1	-	SPM2
135	SCZD	Bonilha L	2008	Schizophr Res 101(1-3):142-51	14	11/3	40±7	3	1	10	SPM5
136	SCZD	Kyriakopoulos M	2009	The British Journal of Psychiatry 195(4): 346-353	17 17	13/4 13/4	16.62±1.28 23.85 ±4.83	1.5 1.5	2.5	4 4	SPM2 SPM2
137	ASD	Ecker C	2012	Arch Gen Psychiatry 69(2): 195-209	89	89/0	26±7	3	1	3	FSL 4.0

138	ASD	Greimel E	2013	Brain Struct Funct 218: 929–942	47	47/0	21.4±10.1	1.5	-	8	SPM5
139	ASD	Riva D	2013	Cerebellum 12: 676–685	26	23/3	5.10±2.6	1.5	5	8	SPM8
140	ASD	Radeloff D	2014	Plos One 9(9): e106539	34	31/3	19.06±5.12	3	1	8	SPM8
141	ASD	D'Mello AM	2015	NeuroImage Clinical 7: 631-9	35	30/5	10.4 ± 1.6	3	-	8	SPM8
142	ASD	Foster NE	2015	Pediatr Neurol 53(4): 350-9	38	38/0	12.4±2.4	3	-	8	CIVET
143	ASD	Osipowicz K	2015	Autism Res 8(4): 379-85	531	430/101	7-64	-	-	8	SPM8
144	ASD	Eilam-Stock T	2016	Front Neurosci 10: 237	66	60/6	27±8	-	-	8	SPM8
145	OCSD	Cheng B	2016	Front Behav Neurosci 10: 141	30	18/22	10.8±1.2	3	1x1.25x2	8	SPM8
146	OCSD	Chen J	2013	Psychiatry Res 213(1): 11-7	8	4/4	11.7±2.7	3	0.5x0.5x1	8	SPM8
147	OCSD	de Wit SJ	2014	Am J Psychiatry 171(3): 340-9	412	202/210	32.10±9.6	1.5	-	10	SPM8
148	OCSD	Hashimoto N	2014	Neuropsychiatr Dis Treat 10: 1987-96	24 15	11/13 7/8	35.70±7.2 32.50±7.7	1.5 1.5	1x1x1 1x1x1	8 8	SPM8 SPM8
149	OCSD	Hou J	2013	PLoS One 8(12): e83931	33	18/15	25.30±9.6	3	1x1x1	8	SPM8
150	OCSD	Huyser C	2013	World J Biol Psychiatry. 14(4): 319-31	29	11/18	13.78±2.58	3	1x1x1.2	8	SPM8
151	OCSD	Kobayashi T	2015	Magn Reson Med Sci 14(4): 329-35	20	10/10	34.1±8.5	3	1.5x1.5x1.5	8	SPM8
152	OCSD	Lazaro L	2014a	Prog Neuropsychopharmacol Biol Psychiatry 54: 249-58	62	36/26	15.4±2.1	3	1x1x1	8	SPM8
153	OCSD	Okada K	2015	Psychiatry Clin Neurosci 69(7): 411-21	37	14/23	34.4±10.5	3	1	8	SPM8
154	OCSD	Park SE	2015	Psychiatry Clin Neurosci 69(11): 717-23	14	9/5	28.9±7.2	3	1	8	SPM8
155	OCSD	Subira M	2013	PLoS One 8(9): e75273	65 30	29/36 20/10	34.6±9.43 32.23±9.05	1.5 1.5	1.2 1.2	8 8	SPM8 SPM8
156	OCSD	Subira M	2015	J Psychiatry Neurosci 40(4): 232-40	71	35/36	32.11±8.45	1.5	1	10	SPM8
157	OCSD	Tan L	2013	Neurosci Bull 29(5): 642-8	28	19/9	25.35±7.24	1.5	1	8	SPM5
158	OCSD	Tang W	2013	Prog Neuropsychopharmacol Biol Psychiatry 46: 126-31	18	11/7	25.5±6.7	3	1	-	SPM8
159	OCSD	Tang W	2015	Behav Brain Res 294: 72-80	26	15/11	25.5±4.9	3	1	-	SPM8
160	OCSD	Tang W	2016	Behav Brain Res 313: 17-22	18	11/7	27.3±10.4	3	1.8	4	SPM8
161	SCZD	Amann BL	2016	Acta Psychiatr Scand 133(1): 23-33	45 45	26/19 26/19	43.2±9.1 43.2±9	1.5 1.5	1 1	- -	FSL-VBM FSL-VBM
162	SCZD	Anderson VM	2015	Int J Neuropsychopharmacol 18(7): pyv016.	18 19 15	14/4 14/5 13/2	32.2±7.9 33.3±8 34.3±7.1	3 3 3	0.8 0.8 0.8	- - -	FSL-VBM FSL-VBM FSL-VBM
163	SCZD	Chen Z	2014	Psychol Med 44(12): 2489-501	86	47/39	24.52±0.91	3	1	6	SPM2
164	SCZD	Filippi M	2014	AJNR Am J Neuroradiol 35(1): 30-7	43	24/19	29.3±7.4	1.5	1	8	SPM8
165	SCZD	Frascarelli M	2015	Psychiatry Res 231(2): 103-10	18	8/10	29.2±8.6	3	1	8	SPM8

					15	9/6	42.7±7.4				
166	SCZD	Fukuta H	2013	Psychiatry Clin Neurosci 67(1): 3-11	40	0/40	45.6±8.1	1.5	1.5	12	SPM8
167	SCZD	Guo Q	2014	Schizophr Res 160(1-3): 57-66	19	3/16	23.95±7.49	3	1	6	SPM8
168	SCZD	Guo W	2015	Medicine (Baltimore) 94(42): e1493	49	30/19	22.69±4.62	1.3	-	-	SPM8
169	SCZD	Huang P	2015	Sci Rep 5: 14505	18 18	10/8 9/9	22.56±6.73 22.67±3.85	3	1	8	SPM8
170	SCZD	Kim GW	2015	Neuroreport 26(18): 1095-100	20	12/8	30±1	3	1	6	SPM8
171	SCZD	Kong L	2014	Psychiatry Res 231(2): 176-83	22	16/6	53.95±8.53	3	1	8	SPM8
172	SCZD	Lei W	2015a	Psychiatry Res 234(2): 219-26	88 44	52/36 26/18	23.04±6.94 22.91±6.89	3 3	1 1	6 6	SPM8 SPM8
173	SCZD	Lei W	2015b	Sci Rep 5: 12994	33	22/11	22.33±6.9	3	1	6	SPM8
174	SCZD	Liao J	2015	J Psychiatr Res 65: 80-6	93	57/36	27±6.6	3	1	8	SPM8
175	SCZD	Kenneth Martin A	2014	Psychiatry Res 224(3): 311-8	26	-	-	3	0.9	8	SPM8
176	SCZD	Nakamura K	2013	Front Psychiatry 4: 16	34	20/14	13.5±2	1.5	1	10	SPM8
177	SCZD	Picado M	2015	PLoS One 10(4) :e0126407	20	11/9	35.9±0.73	1.5	2	12	SPM8
178	SCZD	Poletti S	2016	J Affect Disord 189: 290-7	96	67/29	37.24±9.33	3	0.8	8	SPM8
179	SCZD	Rose EJ	2014	Am J Med Genet B Neuropsychiatr Genet 165B(6): 467-71	163	52/111	38.51±10.84	MULTICENTRE			
180	SCZD	Salgado-Pineda P	2014	Neuropsychobiology 69(1): 52-8	14	9/5	37.29±8.87	3	-	12	SPM5
181	SCZD	Sans-Sansa B	2013	Schizophr Res 146(1-3): 308-13	31	24/7	40.71±8.61	1.5	1	-	FSL-VBM
182	SCZD	Singh S	2014	Neuroradiology 56(5): 413-22	14	8/6	34.06±9.89	3	1	10	SPM8
183	SCZD	Singh S	2015	J Biosci 40(2): 355-64	14	11/3	31.5±9.4	3	1	10	SPM8
184	SCZD	Song J	2015	Neuropsychiatr Dis Treat 11: 1211-9	71	29/42	36.6±14.7	3	1	8	SPM8
185	SCZD	Stegmayer K	2016	Cortex 78: 125-37	45	28/17	38.24±11.37	3	1	8	SPM8
186	SCZD	Stegmayer K	2014	Psychiatry Res 223(1): 49-51	46	29/17	34.7±11.5	3	1	8	SPM8
187	SCZD	Suazo V	2014	Psychiatry Clin Neurosci 68(3): 206-15	17	10/7	33.29±10.48	1.5	1.5	-	SPM8
188	SCZD	Torres US	2016	Neuroimage Clin 12: 1-15	161	111/50	30.4±8.3	MULTICENTRE			
189	SCZD	Vijayakumari AA	2015	Clin Psychopharmacol Neurosci 13(1): 68-82	41	29/12	27.34±7.48	3	1	8	SPM8
190	SCZD	Yao L	2014	MAGMA 27(4): 283-90	68	30/38	24.2±8.6	3	1	4	SPM8
191	SCZD	Yue Y	2016	PLoS One 11(1): e0147204	20	10/10	24.45±5.51	3	1	8	SPM8
192	SCZD	Zhang Y	2015	Schizophr Res 168(1-2): 353-9	37	17/20	15.5±1.8	3	1	8	SPM8
193	SCZD	Horacek J	2012	World J Biol Psychiatry 13(7): 501-9	44	22/22	30.82±9.76	1.5	1	8	SPM5

194	SCZD	Rigucci S	2013	Acta Psychiatr Scand 128(4): 261-70	19	12/7	22.2±3.7	1.5	1	6	SPM8
195	SCZD	Sarrò S	2013	Br J Psychiatry 203(1): 51-7	81	59/22	42.96±9.85	1.5	-	-	FSL-VBM
196	SCZD	Wang Q	2013	Psychological Medicine 43(11): 2301-2309	35	16/19	23.84±6.96	3	3x3x3	6	SPM8 & DTIstudio
197	SCZD	Situ W	2015	International Journal of Psychiatry in Clinical Practice 19(2): 114-118	50	50/0	31.1±6.48	1.5	4x4x4	8	SPM2
198	SCZD	Zhang XY	2016	J Clin Psychiatry 77(2): 205-211	39	16/23	28.87±10.22	3	2.4	6	FSL-FMRIB & SPM8
199	SCZD	Wagner G	2015	Cortex 66: 35-45	38	25/13	35.8±9.9	3	2.5	4	FSL-FMRIB & SPM8
200	OCSD	Admon R	2012	Psychiatry Res 203(2-3): 207-13	13	10/3	25.5±1	1.5	3	8	Matlab DTItool
201	OCSD	Lazaro L	2014b	Depress Anxiety 31(12): 1007-17	63	36/27	15.6±2.1	3	2	8	FSL-FMRIB & SPM8
202	OCSD	Li Z	2014	Med Sci Monit 20: 2275-82	11 11	- -	18-65 18-65	3 3	3 3	8 8	FSL-FMRIB & SPM8 FSL-FMRIB & SPM8
203	OCSD	Gruner P	2012	Neuropsychopharmacology 37(12): 2730-9	30	13/10	14.3±2.1	3	2.5	8	FSL-FMRIB & SPM5

* The average age (standard deviation) or age range is reported on the basis of what is specified by the authors.

** Where no information was provided, the voxel-size was expressed.

Table S2. Description of the total experimental sample with the respective diagnostic labeling.

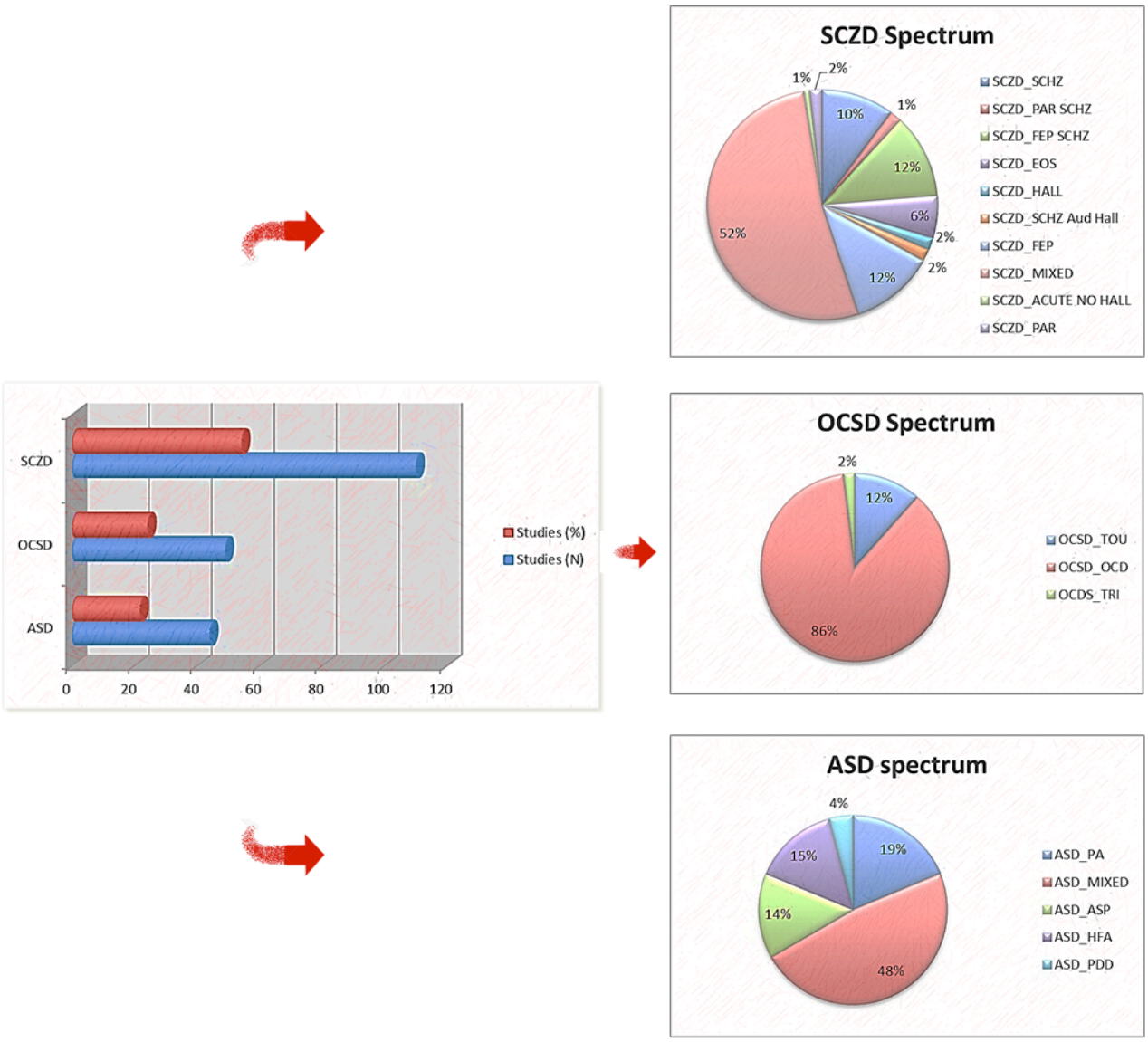
SPECTRUM	N	DIAGNOSTIC SUBCATEGORIES (N)*	MALE/FEMALE**	AGE (MEAN +SD) **
ASD	1719	1172 Mixed form	931/200	16.858 ± 10.226
		200 Primary Autism	172/28	15.166 ± 10.625
		171 High-functionality	156/15	15.363 ± 10.126
		134 Asperger	94/25	23.483 ± 10.607
		42 Pervasive Development Disorder	40/2	21.750 ± 10.349
OCSD	1738	1584 Obsessive-Compulsive Disorder	806/43	25.871 ± 9.854
		136 Tourette Syndrome	120/16	28.358 ± 10.272
		18 Trichotillomania	1/17	37.39 ± 11.65
SCZD	5236	3186 Mixed form	1994/1124	33.741 ± 9.639
		673 Schizophrenia Simplex	388/285	36.098 ± 9.215
		565 First Episode Schizophrenia	288/236	25.176 ± 9.875
		449 First Episode Psychosis	240/160	23.626 ± 8.900
		184 Early Onset Symptoms of Psychosis	111/73	16.218 ± 9.018
		49 Paranoia	28/21	32.400 ± 8.950
		48 Paranoia with Schizophrenia symptoms	26/22	35.645 ± 9.522
		39 Auditory Hallucination	10/8	30.780 ± 8.987
		30 Hallucination	25/5	24.755 ± 8.945
		13 Acute Psychosis with no hallucination	8/5	31.000 ± 9.300

* Diagnoses are reported on the basis of what is specified by the authors.
** Values have been calculated on the basis of the studies bearing the required information.

ASD = Autism Spectrum Disorder
OCSD = Obsessive Compulsive Spectrum Disorder
SCZD = Schizophrenia Spectrum Disorder

N = number of subjects from each diagnostic category

Figure S2. Statistical distribution of the papers included in the study.



Legend

SCZD_SCHZ = Schizophrenia Simplex; SCZD_PAR SCHZ = Paranoia with Schizophrenia symptoms; SCZD_FEP SCHZ = First Episode Schizophrenia; SCZD_EOS = Early Onset Symptoms of Psychosis; SCZD_HALL = Hallucination; SCZD_Aud Hall = Auditory Hallucination; SCZD_FEP = First Episode Psychosis; SCZD_MIXED = Mixed form; SCZD_ACUTE NO HALL = Acute Psychosis with no hallucination; SCZD_PAR = paranoia; OCSD_TOU = Tourette Syndrome; OCSD_OCD = Obsessive-Compulsive Disorder; OCSD_TRI = Trichotillomania; ASD_MIXED = Mixed form; ASD_PA = Primary Autism; ASD_HFA = High-functionality; ASD_ASP = Asperger; ASD_PDD = Pervasive Development Disorder.

Table S4[2]. Gray matter (GM) and white matter (WM) variations with relative numbers of foci for each of the selected psychiatric spectra.

SPECTRUM	*Diagnostic Label*	*Foci (N)*	*White matter changes*		*Gray matter Changes*	
			⇑	⇓	⇑	⇓
ASD (N=1719)	*Mixed form*	312	27	66	125	94
	Primary autism	186	7	57	93	29
	Asperger	54	11	14	2	27
	High-functionality autism	53	5	20	16	12
	Pervasive developmental disorder	16	0	11	0	5
OCSD (N=1738)	*Obsessive-compulsive disorder*	364	25	77	67	195
	Tourette syndrome	70	13	33	5	19
	Trichotillomania	4	0	4	0	0
SCZD (N=5236)	*Schizophrenia simplex*	190	0	4	4	182
	Paranoia	20	0	17	1	2
	Auditory hallucination	34	7	0	0	27
	First episode psychosis	92	3	45	4	40
	Mixed form	633	18	160	22	433
	First episode schizophrenia	65	5	19	0	41
	Acute psychosis with no hallucination	21	0	21	0	0
	Early onset symptoms of psychosis	52	0	8	0	44
	Hallucination	21	0	16	0	5
	Paranoia with schizophrenia symptoms	26	0	9	1	16
		Foci	121	581	340	1171
		(Total)	702		1511	

[2] The items shown in the table are the result of the entire selection process as shown in PRISMA (2009) flow chart and table S2. The starting point for the selection can be traced in the algorithms and in the additional considerations previously proposed.

The insular nodes and their resting state functional connectivity

Subjects and image acquisition

For the anatomical covariance and functional connectivity measures we used the Beijing dataset which has been publicly released within the "1000 Functional Connectomes" Project. This dataset consists of 198 subjects (76 males and 122 female) with age ranging from 18 to 26 years, mean 21.16, SD 1.83, that underwent structural and resting state scans. All subjects were right-handed and had no history of neurological or psychiatric disorders. Written informed consent was obtained from each participant, and the study was approved by the Institutional Review Board of Beijing Normal University Imaging Center for Brain Research.

MRI data were acquired using a SIEMENS TRIO 3-Tesla scanner in the Beijing Normal University Imaging Center for Brain Research. Participants lay supine with their head fixed by straps and foam pads so as to minimize movements. During the resting-state session, participants were instructed to be as still as possible and let their mind roam. Functional images were obtained using an EPI sequence with the following parameters: 33 axial slices, thickness/gap = 3/0.6 mm, in-plane resolution = 64 × 64, TR = 2000 ms, TE = 30 ms, flip angle = 90°, FOV = 200 × 200 mm. Furthermore, a T1-weighted sagittal three-dimensional magnetization-prepared rapid gradient echo (MPRAGE) sequence was acquired, which covered the entire brain: 128 slices, TR = 2530 ms, TE = 3.39 ms, slice thickness = 1.33 mm, flip angle = 7°, inversion time = 1100 ms, FOV = 256 mm × 256 mm, and in-plane resolution = 256 × 192.

Preprocessing

In order to analyze the functional connectivity of the insula we used the DPABI processing tool rel. 2.3 (Yan, et al., 2016). All the preprocessing was performed by employing the advanced DPARSF module V3.2. The first 10 volumes of the functional images are often discarded both for achieving signal equilibrium and for letting participants adapt to the scanning noise. All volume slices were corrected for different signal acquisition times taking the middle slice as reference and using the odd slice order. Then, the images' time series of every subject were realigned. After the realignment, individual structural images were co-registered to the mean functional image. The transformed structural images were then segmented into GM, WM and CSF. To remove the nuisance signals, we employed the Friston 24-parameter model (Friston, et al., 1996), three translation and three rotation parameters of the current volume and the preceding volume, plus each of these values squared, so as to regress out head motion effects from the realigned data. The signals from both WM and CSF were regressed out to reduce respiratory and cardiac effects. In addition, linear and quadratic trends were also included as regressors, since the BOLD signal exhibits low-frequency drifts. The DARTEL tool was used to transform the functional data from individual native space to MNI space. Spatial smoothing (FWMH kernel: 4.5 mm) was applied to the functional images and temporal filtering (0.01–0.1 Hz) was then performed on the time series. Finally, for every subject the mean time course of every insular ROI was extracted and correlated voxel-wise to all the voxels of the brain. These results were then summarized using a one sample t test. All the calculations were thresholded at a $p<0.05$ cluster-level, corrected for multiple comparison using the FSL randomize tool.

Clustering and visualization of alteration patterns

Clustering

To test if data can be decomposed in two or more groups we employed a clustering approach: the k-means technique. This technique is an unsupervised learning algorithm that subdivides a set of objects in k groups according to their attributes. There are different types and methods of clustering in literature: we chose k-means because, compared to other algorithms, its runtime and performance are usually more efficient as the number of records increases (Bishop, 2006). Furthermore, since clusters are non-hierarchical, they do not overlap, which is important in our case, as we were looking for a clear partition between the three psychiatric spectra (Bishop, 2006; Thirion, et al., 2014).

For the k-means clustering we used an nxp matrix, in which rows correspond to points and columns to variables (attributes). We used as attributes the MA maps and as points the voxels. The optimal number of groups (k) was determined by the silhouette plot introduced by Kaufman and Rousseeuw (1990). The results of the silhouette plot showed that 2 was the best number of clusters calculated by this algorithm (Fig. S3).

In this study we applied both clustering and classification methods to analyze our data. The rationales for using the clustering technique as well as the machine learning method are different. We used the clustering technique to check if a "natural" decomposition of the voxels could emerge from each different psychiatric disorder. This allowed us to see whether or not there were interesting anatomical partitions that could differentiate ASD, SCZD, and OCSD.

Multidimensional scaling

To evaluate the similarity between the three different spectra of psychiatric disorders (represented by MA maps generated by the different experiments analyzed in this study) we used the multidimensional scaling (MDS) method. MDS mainly consists of data proximity analysis techniques to check hidden structures. For each paper, every MA map was transformed into a series of vectors containing all the values of the original matrix. Then a representational similarity matrix was constructed by computing the correlation r among all vectors. Vectors are constituted by the statistical values of the voxels pertaining to the MA maps. The distance matrix (or representational dissimilarity matrix) defined as $1 - r$ (Cauda, et al., 2014; Kriegeskorte, et al., 2008) was similarly created. The distance matrix was subjected to the multidimensional scaling analysis so as to obtain a geometrical representation of data deviation.

We evaluated the similarity by examining visually the MDS graphs, in which the multidimensional similarity/dissimilarity between pathology-derived MA maps are represented as 2D distances (expressed in arbitrary values). Similar pairs are placed close together and dissimilar pairs are placed far between each other (Fig. S3).

Figure S3. Left panel: Clustered gray matter (GM) distance matrices of the modeled activation (MA) maps relative to each of the examined experiments. Right panel: multidimensional scaling (MDS) of the modeled activation (MA) maps relative to each of the examined experiments (graphs

expressed in arbitrary units). In MDS graphs the multidimensional similarity/dissimilarity between pathology-derived MA maps are represented as 2D distances (expressed in arbitrary values). Similar pairs are placed close together and dissimilar pairs are placed far between each other.

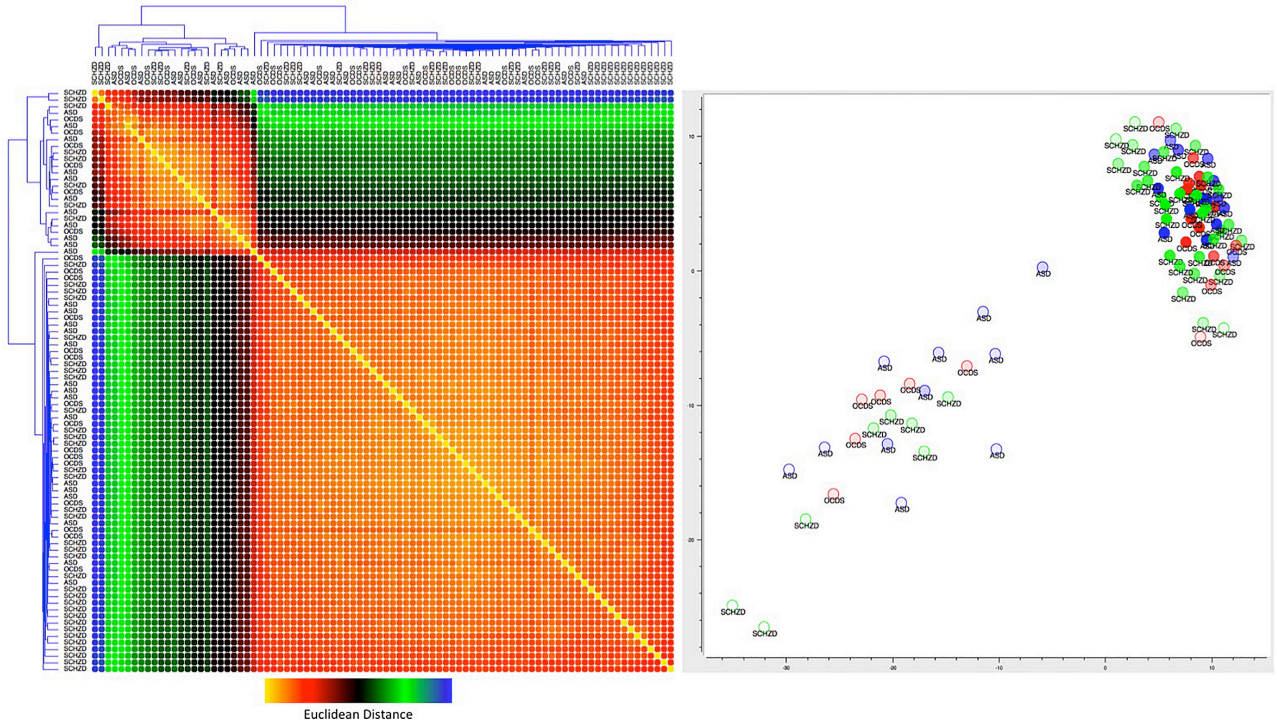
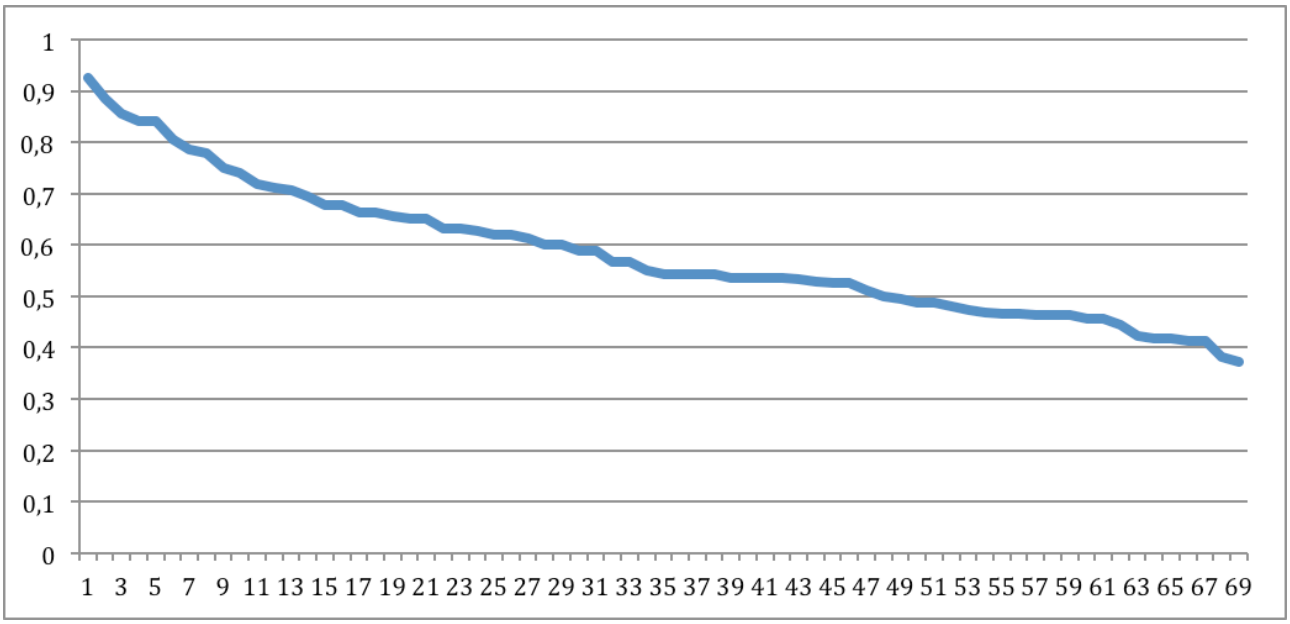


Figure S4. Patel's k values for the 70 co-atrophy edges.



Schizophrenia vs the whole dataset

The results obtained with the analysis of schizophrenia data only are very similar to those obtained with the analysis of the whole dataset, in which all the three spectra are taken into consideration (Fig. S5-S6). Changes regard in particular Patel's k values of single edges, but the two networks are substantially very similar. These results are consistent with those illustrated in Figure S3, which shows that the first cluster (encompassing all the three spectra) represents the most part of our data.

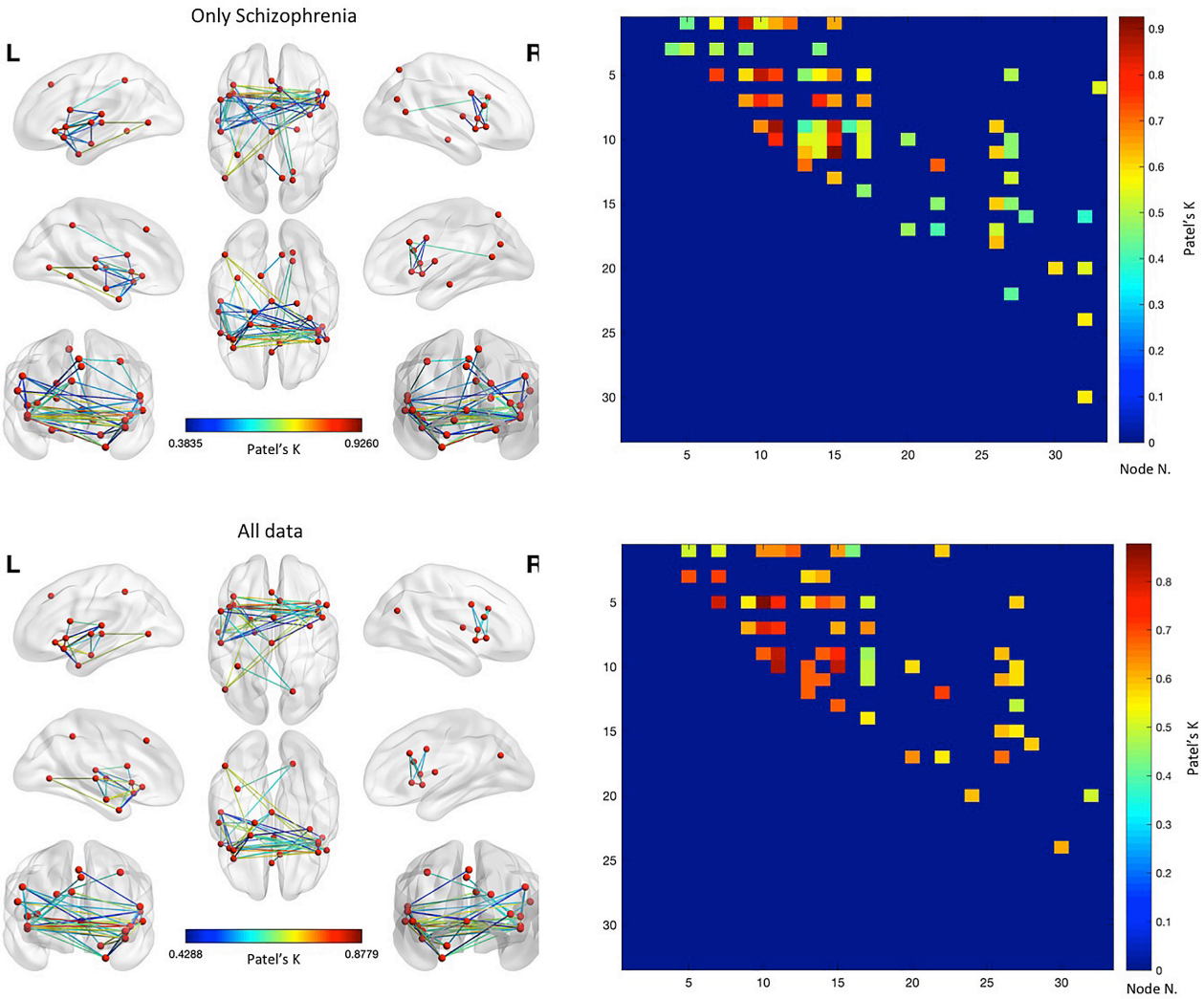


Figure S5. Results of the morphometric co-atrophy network constructed with schizophrenia data only or with the whole dataset. Colors from blue to red indicate increasing Patel's k values (i.e., increasing co-alteration probabilities).

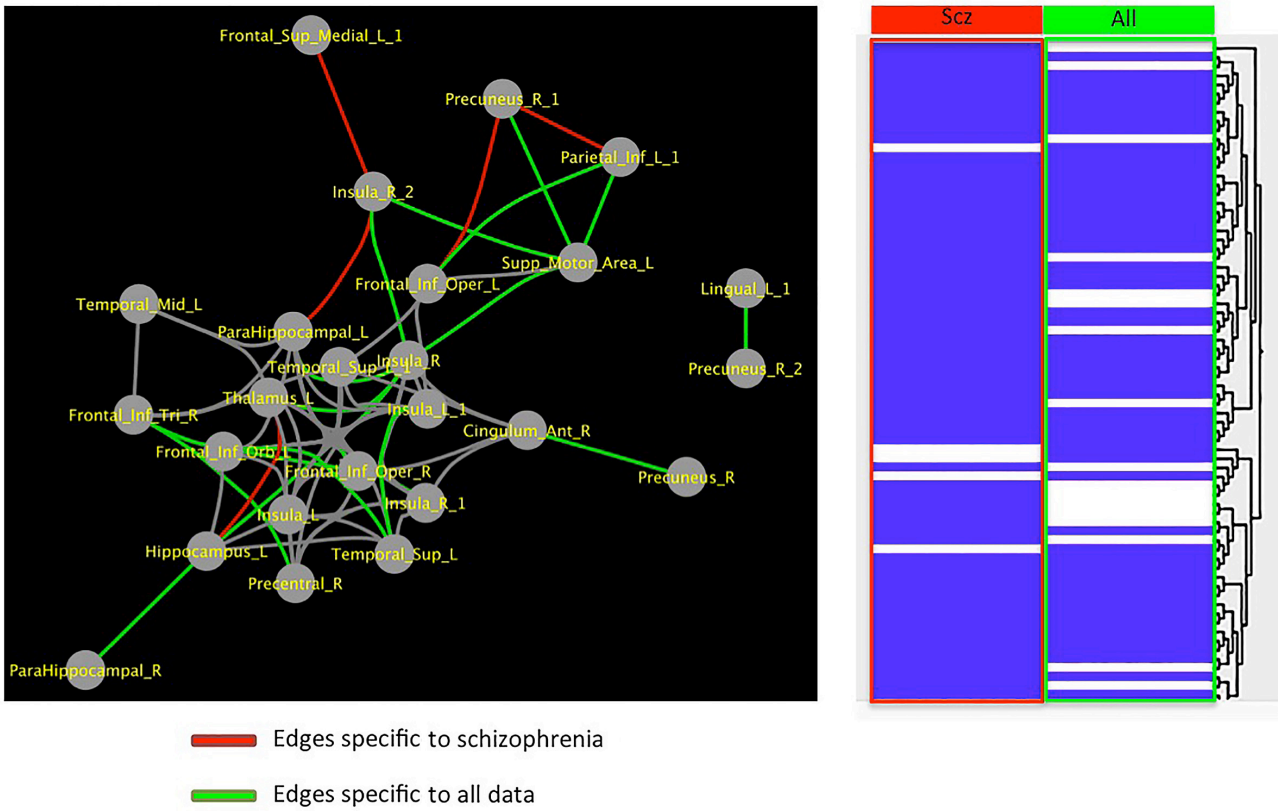


Figure S6. Results of the morphometric co-atrophy network constructed with schizophrenia data only or with the whole dataset. The left panel shows the differences between the results. Red edges are specific to schizophrenia, while green edges are specific to all the three spectra. The right panel shows the edges clustered by spatial distance. White lines indicate absence of edges, blue line their presence.

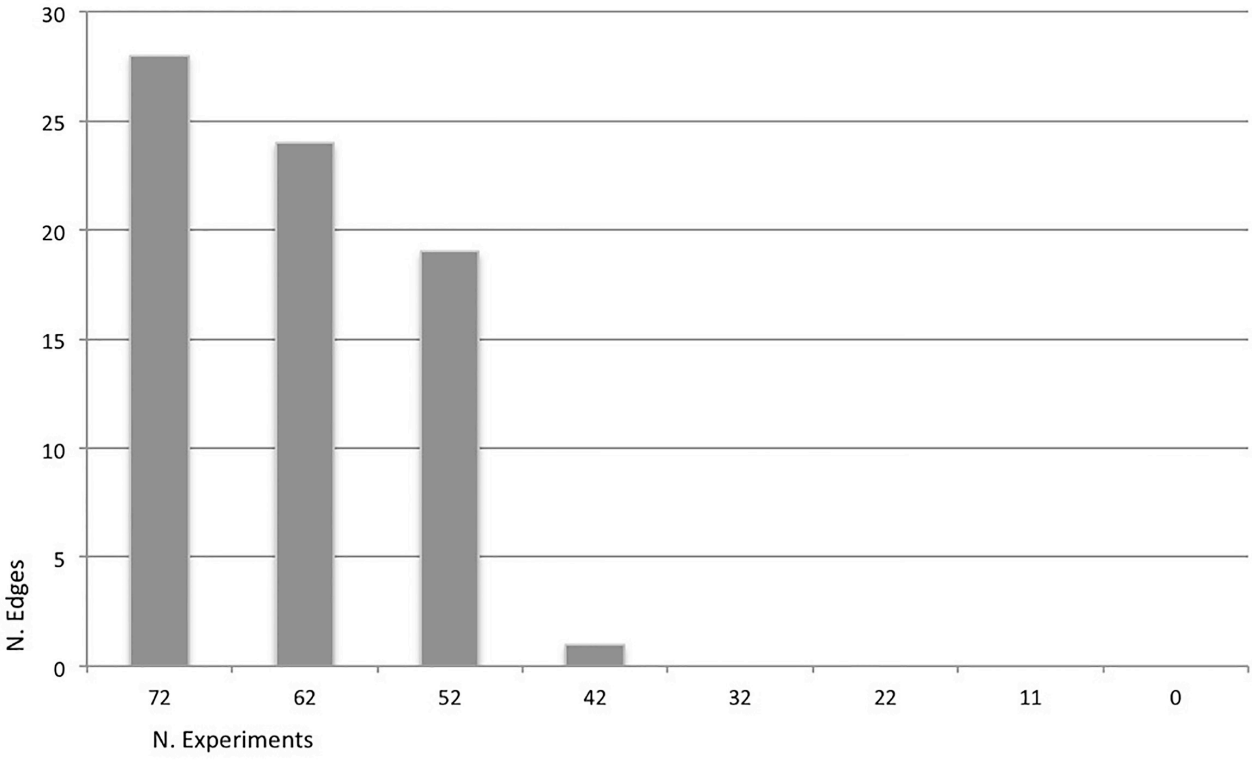


Figure S7. Relationship between experiments and edges in the Schizophrenia dataset. The figure illustrates how the results of the statistical analysis change if 10 experiments are removed from the SCZD sample at each simulation. With this procedure we were able to evaluate the variation of the number of significant edges estimated by our statistics. It is clear that the amount of edges decreases after every removal: after 50 experiments the number of significant edges collapses and after 40 experiments no edge can be identified.

References

Bishop, C. (2006) Pattern Recognition and Machine Learning. Springer-Verlag New York.

Cauda, F., Costa, T., Palermo, S., D'Agata, F., Diano, M., Bianco, F., Duca, S., Keller, R. (2014) Concordance of white matter and gray matter abnormalities in autism spectrum disorders: A voxel-based meta-analysis study. Human brain mapping, 35:2073-98.

Friston, K.J., Williams, S., Howard, R., Frackowiak, R.S., Turner, R. (1996) Movement-related effects in fMRI time-series. Magnetic resonance in medicine : official journal of the Society of Magnetic Resonance in Medicine / Society of Magnetic Resonance in Medicine, 35:346-55.

Jaccard, P. (1901) Étude comparative de la distribution florale dans une portion des Alpes et des Jura. Bulletin de la Société Vaudoise des Sciences Naturelles, 37:547-579.

Kaufman, L., Rousseeuw, P.J. (1990) Finding groups in data. Wiley, Hoboken, New Jersey.

Kriegeskorte, N., Mur, M., Bandettini, P. (2008) Representational similarity analysis - connecting the branches of systems neuroscience. Frontiers in systems neuroscience, 2:4.

Thirion, B., Varoquaux, G., Dohmatob, E., Poline, J.B. (2014) Which fMRI clustering gives good brain parcellations? Frontiers in neuroscience, 8:167.

Yan, C.-G., Wang, X.-D., Zuo, X.-N., Zang, Y.-F. (2016) DPABI: Data Processing & Analysis for (Resting-State) Brain Imaging. Neuroinformatics, 14:339-351.